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An Evaluation of a Multi-Component Intervention for Loud Speech in Children with Autism Spectrum Disorder

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Dedication

This dissertation is dedicated to the family members, friends, and teachers who believed in me, nudged me forward, listened to my worries and complaints, and cheered for me when it was all finished. You’re the real MVPs.

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An Evaluation of a Multi-Component Intervention for Loud Speech in Children With Autism Spectrum Disorder

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Idiosyncratic patterns of speech are common in ASD and greatly affect an individual's level of functioning, and as a result, the extent of their social and educational inclusion. Although there is a large body of literature detailing and evaluating interventions for a variety of verbal behaviors in ASD, there is a relative dearth of research describing interventions for idiosyncratic characteristics of communicative speech (e.g., atypical prosody) and even less focused specifically on loud speech. To address this gap in the literature, the current study presents and evaluates a treatment package implemented with three children with ASD and a history of loud speech (i.e., ≥ 70 db). A concurrent multiple baselines across participants design was used to determine whether a multi-component intervention (i.e., an antecedent modification, a differential reinforcement of other behavior (DRO) procedure, and in-vivo feedback) effectively reduced participants' rates of loud speech. The results suggest it is possible to decrease rates of loud speech in children with ASD to near-zero levels by consistently implementing a relatively simple combination of behavioral strategies. The present study

extends the literature on speech prosody in ASD, and fills a gap in the treatment literature by detailing an effective intervention for loud speech. This research could also inform future investigations into this nuanced yet crucial aspect of social communication, including appropriate methods for addressing issues with speech loudness in individuals with ASD.

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Chapter One: Introduction

According to the latest edition of the Diagnostic and Statistical Manual (DSM-5), autism spectrum disorder (ASD) is a complex neurodevelopmental disorder characterized by: (1) persistent difficulties in social communication, and (2) repetitive or restrictive patterns of behaviors (American Psychological Association, 2013). To meet criteria for diagnosis, individuals must demonstrate deficits in social communication across multiple contexts (e.g., home, work, school, community settings). Examples of these deficits include difficulty understanding social-emotional reciprocity, initiating and maintaining conversations, and developing or understanding relationships. Similarly, the individual must also exhibit two or more of the following restricted and repetitive behaviors: stereotyped or repetitive motor movements, insistence on sameness or inflexible adherence to routines, highly restricted and fixated interests, or hyper/hyporeactivity to certain sensory stimuli.

Prevalence and Cost of ASD

Although prevalence estimates for ASD have been steadily increasing over the past 30 years, results from a large (N = 363,749) nationwide study conducted by the Centers for Disease Control (CDC) in 2014 suggest that ASD currently affects around 1 in 68 children in the United States, and is about five times more common among boys (1 in 42) than girls (1 in 189). These numbers nearly double the estimated prevalence of 1 in 150 just a decade earlier (CDC, 2002). It is unclear why the prevalence rates of ASD have continued to rise, but most experts attribute the trend, in part, to improvements in diagnostic criteria and assessment instruments, as well as increased awareness and recognition of the disorder among practitioners and the general public. Still, these factors alone do not sufficiently explain the upward trend in ASD's prevalence. It is therefore possible that individuals born today are truly more likely to have ASD than individuals born in previous decades.

Based on estimates of the average lifetime cost of care for one individual with ASD, researchers have determined that this disorder costs the nation approximately $137 billion each year (Mandell & Knapp, 2011)—a number calculated using a previous estimated prevalence rate of 1 in 110 (CDC, 2009), which suggests that the figure is almost certainly an underestimate. Taken together, it is apparent that ASD has become a public health crisis with a staggering cost to society. Not surprisingly, the rising prevalence of ASD has generated a corresponding increase in ASD research (see Singh, Illes, Lazzeroni, & Hallmayer, 2009) as scientists, practitioners, and parents seek answers about potential causes of and treatments for ASD.

Etiology of ASD

Etiologically, current research suggests that ASD can be linked to both genetic and environmental influences (Boyle et al., 2011; Hallmayer et al., 2011). There is a growing body of evidence indicating that the heritability of autism is extremely complex, and may involve hundreds of genes as well as rare point mutations and chromosomal abnormalities (Lin et al., 2012; Talkowski, Minikel, & Gusella, 2014). Several independent studies have found evidence indicating that ASD may be present and detectable very early in development, perhaps even prenatally in some cases. For example, preliminary research by Allen and Brinster (2014) comparing prenatal ultrasounds of neurotypical children and those who were later diagnosed with ASD suggests that children from the ASD group tended to have noticeably smaller cerebellums at 20 weeks gestation. However, correlational data also indicates that certain environmental factors, such as living in areas with high levels of air pollution (Volk, Lurmann, Penfold, Hertz-Picciotto, & McConnell, 2013), are associated with higher rates of ASD diagnoses. These results suggest exposure to certain toxins or other environmental stressors may precipitate the development of ASD, thus raising concerns that epigenetic influences may exacerbate atypical development in individuals who are predisposed to the disorder. It is possible, therefore, that ASD results from some combination of or interaction between potential causal factors, including genetic predisposition and environmental triggers post-conception.

Heterogeneity of ASD

Unfortunately, there is presently no definitive test for ASD; diagnoses are made on the basis of observable behavioral symptoms, and often not until age 4 or later (CDC, 2012). Because there are so many factors that have been linked to the development of ASD, it has become clear that the disorder can be manifested in a multitude of ways, each looking unique to some degree. As a result, individuals with ASD comprise a heterogeneous group with widely varying degrees of impairment, ranging from mild social challenges to severe intellectual disability.

Level of Functioning

Severity of the disorder is based on the degree of impairment of daily living that results from restricted or repetitive patterns of behavior and difficulties in social communication. More specifically, level of impairment can be categorized as mild (i.e., Level 1- “requiring support”), to severe (i.e., Level 3- “Requiring Very Substantial Support”). Thus, some individuals with ASD are able to live independently, pursue professional careers, and raise children. These individuals require minimal assistance, such as highly structured schedules, visual reminders, or brief verbal explanations, to successfully complete more complex tasks of daily living (e.g., caring for children, completing work assignments). Other individuals, however, may never be able to live independently, and may require extensive support from the environment to complete even the most basic tasks of daily living (e.g., dressing, feeding). In consideration of other factors that may impact an individual’s degree of impairment, diagnosing clinicians must also specify if the disorder occurs with or without accompanying intellectual impairment, language impairment, or catatonia, or is associated with a known medical or genetic condition or environmental cause.

Role of Verbal Behavior in Level of Functioning

One of the core factors that affect the extent to which an individual with ASD is able to live, work, and benefit from a general education setting is his or her ability to communicate effectively. Put simply, the ability to spontaneously communicate needs and desires is essential to social functioning. Because research suggests that ASD is present early in development, it follows logically that the presence of the disorder likely influences speech during early development, perhaps even from birth. Approximately 25-40% of individuals with ASD are nonverbal, meaning they are unable to communicate vocally with speech. The remaining 60-75% often experience a delayed onset of speech, as well as difficulties using speech effectively to communicate.

Idiosyncratic vocal behaviors are common among individuals with ASD and can be categorized under either of the two core criteria used to diagnose the disorder. For example, vocal stereotypy (e.g., echolalia, palilalia) is typically categorized diagnostically as a repetitive behavior and is maintained automatically. Stereotypic utterances are described as ‘vocal’ rather than ‘verbal’ because although they involve speech, they typically do not serve a communicative function. In contrast, purposeful verbal communication is social in nature and maintained by a social function; therefore deficits in this type of verbal behavior are diagnostically considered impairments in social communication.

Because stereotypic vocal behaviors can be disruptive and interfere with learning and socialization, a number of studies have described treatments for these behaviors. For example, a study by Graff, Lineman, Libby, and Aheard (1999) assessed the effects of a consequence-based intervention on the stereotypic screaming of a 6-year-old child with ASD. As predicted, the authors found that the implementation of a 2-min timeout procedure immediately following the occurrence of a scream effectively reduced the behavior to near-zero rates. In another example, Manning and Katz (1991) used peer modeling to reduce echolalia and increase functional communication in an 11-year-old boy with ASD. Notably, the majority of studies describing interventions for idiosyncratic vocalizations have used behavioral strategies to treat the target behavior, which is consistent with myriad evidence that behavioral interventions appear to be best practice for the treatment of challenging behaviors in ASD.

Despite the relatively large body of literature detailing and evaluating interventions for repetitive and restrictive vocal behaviors in ASD, there is comparably much less literature on interventions for idiosyncratic characteristics of communicative speech, and virtually none examining inappropriate speech volume. The dearth of research in this area is concerning, given that strong skills in communicative speech may render the presence of stereotyped verbal behaviors irrelevant. That is, it may be more important to bolster and shape functional speech than to extinguish the presence of non-functional speech, as the presence of the first may diminish the latter.

Chapter Two: Method

Screening and Selection of Participants

To be included in the proposed study, each participant was required to have a prior diagnosis of an ASD that was made by a physician, psychologist, or neuropsychologist using the diagnostic tools standard to their field. During the selection phase, participants were also required to engage in loud speech during at least 10% of speaking opportunities. Duration recording was used to measure the target behavior over the course of one 10-min screening session. A preferred adult (i.e., parent or behavioral therapist) was present at the screening session, and the child had noncontingent access to a variety of toys and games. To ensure that all study participants had a sufficient degree of verbal proficiency, each child was also required to demonstrate basic conversational ability during play, as reported by adults (e.g., parents, therapists) who were familiar with the child.

Children were excluded from participating in the study if any non-communicative vocalizations, such as vocal stereotypy, occurred during the screening session. A vocalization was designated as a *verbal utterance* if it could be classified as a mand (i.e., request), tact (i.e., comment), intraverbal (i.e., verbal response to another's verbalization), or sound intended to communicate meaning (e.g., saying "whoosh" while making a toy airplane fly). Any other vocalization was classified as a *nonverbal utterance*. Children were also excluded from participating in the study if they had a history of seizures, or had been diagnosed with a traumatic brain injury or tic disorder. Participant gender was not used as a criterion for selection.

Participants

Four children, volunteered by their parents and between the ages of 5 and 9 years old, participated in the study. Three of the participants were recruited from a large ABA therapy provider in central Texas where they were receiving comprehensive behavioral interventions to increase skill acquisition and decrease problem behavior. The fourth participant was recruited by word of mouth. As compensation for their time and travel, participants received treatment services free of charge while the intervention under study was being implemented.

Participant 1: Luke

Luke was an 8-year-old male referred to the study by a clinician who worked with him and believed he would benefit from a more focused intervention for loud speech. Prior to his participation in the study, his speech loudness had been targeted informally through interventions created by his parents, including signs posted in their home about expectations with regards to speech loudness as well as a symbolic picture sometimes shown to the child when quieter speech was desired. According to his mother, his loud speech was especially problematic during his downtime at home, and often prevented his younger sibling from being able to take naps in the afternoon.

Participant 2: Amy

The second participant was a 7-year old female who was referred to the study by the supervisor of her behavioral therapy. She had never received treatment for speech loudness prior to her participation in the study. According to her mother and therapists, her speech loudness was consistently high across activities and appeared to be impacting her ability to develop lasting social relationships.

Participant 3: Ben

The third participant was a 5-year-old male who was referred to the study by the supervisor of his behavioral therapy. He had never received treatment for speech loudness prior to his participation in the study. According to his grandmother and therapists, his speech loudness was high across activities at school and during preferred activities with adults outside of school.

Participant 4: Manuel

The fourth participant was a 9-year-old male who was referred to the study by the supervisor of his behavioral therapy. He had never received treatment for speech loudness prior to his participation in the study. According to his mother, his speech loudness was high during preferred activities both in and outside of school.

Setting and Materials

Intervention settings varied for each child, and were selected based on parent reports of settings where loud speech appeared to be problematic. Luke, Amy, and Manuel were seen at their homes, whereas Ben was seen at the clinic where he received ABA therapy. During both intervention and baseline phases, at least one preferred adult was present during each session and a variety of preferred play activities were offered. The preferred adults were behavioral therapists who worked frequently with the child (Amy, Ben, and Manuel), or the child's parent (Luke). The presence of preferred individuals and activities was intended to serve as establishing operations to increase the reinforcing qualities of social communication. Attention was provided noncontingently during all sessions.

All sessions were video recorded using a Flip Video Ultra HD. A digital sound level meter designed for tablets (i.e., SPLnFFT Noise Meter) was used to measure and record the speech loudness of all speakers for each session. Previous research has suggested this sound level meter yields valid and reliable results across frequencies in a controlled setting (Nast, Speer, & Le Prell, 2014). Data from the sound level meter were coded according to speaker (e.g. participant, preferred adult, study investigator) to determine the frequency of the participant's loud speech. To ensure reliability of its measurement, the sound level meter was calibrated once using a sound level calibrator before the start of the study, which is consistent with professional standards for reliable data collection (SP Technical Institute of Sweden, 2014).

Experimental Design

A concurrent multiple baseline design across participants with an embedded reversal design was used to evaluate the effectiveness of a behavioral treatment for loud conversational speech in children with autism spectrum disorders (ASD). Within the multiple baseline design, following baseline a multi-component intervention was implemented, which included the use of a differential reinforcement of other behavior (DRO) procedure, an antecedent-based modification, and a consequence (i.e., verbal reminder) for undesired behavior, would produce decreases in loud speech (i.e., ≥ 70 dB) during conversations with adults during preferred activities.

Study Phases

The study was carried out in four phases: baseline, intervention phase 1, reversal to baseline, and intervention phase 2. The initiation and termination of each phase was determined by visual analysis of the data, including level, trend, and variability. Collection of baseline data for all participants began in the same week, and study sessions were conducted 1-2 times per week for each participant. Each session lasted approximately 5 minutes, with a maximum of five sessions occurring in a single day and at least one study investigator present for all sessions.

Procedures

The play activity was selected by the participant prior to entering the target setting, and varied across participants and sessions. Examples of activities selected by participants included board games, imaginary/role play, and card games. For each session, the preferred adult's role was to facilitate play and conversation between him or her and the participant. To facilitate these interactions, the adult was instructed to ask questions, make statements, use affective cues (e.g., smiling broadly in response to the child's statement), and provide partial verbal prompts when necessary. Upon entering the target setting, the study investigator and the child's therapist remained within 5 ft. of the child throughout the duration of the session to allow for accurate measurement with the sound level meter. Any instances of challenging behavior were managed according to the child's usual behavior intervention plan.

Baseline. During the baseline phase, participants received no specialized instructions or reminders regarding behavioral expectations prior to entering the target setting. Similarly, no consequences were provided in this phase for either appropriate or inappropriate speaking volume.

Intervention. In the intervention phase, an antecedent-based modification was first implemented prior to entering the target setting (e.g., classroom, living room). Specifically, the investigator reminded the child that he or she should use an inside voice during the following activity and could earn a pre-selected reinforcer for doing so (i.e., "[Name], remember to use an inside voice while you play [activity]. If you do, you can earn [reinforcer]."). Participants were also reminded before the first session in each treatment phase that this meant another person should be able to hear them if they are playing or talking together, but people in another room should not be able to hear them. Reinforcers were chosen at the beginning of each day of data collection through a multiple stimulus without replacement (MSWO) preference assessment (DeLeon & Iwata, 1996). If a participant engaged in loud speech after entering the target setting, the investigator signaled to the child's therapist or parent that loud speech had occurred, and the child immediately received a verbal reminder from the therapist or parent to use an inside voice in order to earn the chosen reinforcer. If the participant engaged in speech of an appropriate volume for 1 min, the investigator signaled to the child's therapist or parent that criteria for reinforcement had been met, and the child immediately received a verbal notification from the therapist or parent that reinforcement had been earned. For Ben, this reinforcement was a small, highly preferred edible (i.e., one Skittle) that could only be earned during study sessions. For Luke, Amy, and Manuel, this reinforcement was a single "point" that could be traded for preferred items or activities after sessions as part of a token economy. Because these three participants were all using token economies prior to their participation in the study, token training was not conducted.

Therapist/Caregiver Training

Therapist/caregiver training on the implementation of baseline and treatment phases were conducted for 10 min prior to the first session of each phase. During these trainings, the therapist/caregiver was given a handout with written instructions as well as operational definitions for loud speech. Investigators read through the written descriptions of procedures while modeling them with another investigator or the therapist/caregiver. Then, each therapist/caregiver was asked to role-play the procedures with the investigator. During the role-play, one investigator pretended to be the child, while the therapist/caregiver practiced implementing the study procedures. Immediately following this training, the therapist/caregiver began conducting sessions. During sessions, the investigator monitored the sound level meter and delivered *in vivo* instructions to therapist/caregiver (e.g., 'give the verbal reminder', 'provide reinforcement') when the target behaviors (e.g., one occurrence of loud speech or 1-min of appropriate speech) were observed or when therapists ask questions.

Data Collection

All sessions were video recorded. Investigators also recorded the target behavior over the course of 5-min sessions using a sound level meter and coded these data after each day's sessions. For the baseline phases, data were collected only on the child's speaking volume. During intervention phases, data were also collected on accuracy of the therapist's implementation of the study protocol (i.e., provision of vocal reminders of expectations for speech loudness prior to entering target setting, provision of consequences following occurrence or nonoccurrence of target behavior). Thus, data on the child's target behavior (i.e., speech volume) were collected for all sessions across all study phases, whereas data on the therapist's or caregiver's behavior were collected for all sessions during only the intervention phases.

Event recording was used to assess each participant's engagement in loud speech, defined as speech greater than or equal to 70 dB, throughout each session. Trained reviewers coded video data to determine the total frequency of loud speech for each session, which was then converted to a rate (i.e., occurrences of loud speech per minute). Similarly, these recordings were also used to categorize participants' verbal behavior (i.e., *verbal utterance* or *nonverbal utterance*) and assess treatment integrity. Vocalizations that were categorized as *nonverbal utterances* were not included in the data analyses with regards to speech loudness.

Interobserver Agreement and Treatment Integrity

A second observer coded video data for 8 sessions per participant, or 32% of all sessions. For each participant, one session from each study phase was randomly selected for coding by a second observer. The final 4 sessions to be coded for each participant were randomly selected from the remaining sessions without regard to study phase. This approach was chosen to ensure that at least one session per study phase was coded by a second observer for each participant.

Interobserver agreement (IOA) for the dependent variable (i.e., rates of loud speech) was calculated by dividing each session into successive 10-s intervals and agreement percentages were determined on an interval-by-interval basis. For example, if one rater recorded 3 occurrences of loud speech for a given interval, whereas a second rater recorded only 2 occurrences of loud speech for that interval, their agreement for the interval would be 67%. The resulting interval agreement percentages were then averaged for each session. Table 1 depicts the mean agreement for each participant as well as the group. Average interobserver agreement across sessions was 97.88%. The agreement was 100% for Luke, 92.6% to 100% for Amy, and 81.4% to 100% for Ben.

To ensure the proposed intervention was implemented as designed, the extent to which therapists correctly adhered to the study protocol (i.e., *treatment integrity*) was also measured. Two observers independently coded recordings of three randomly selected intervention sessions for each participant and completed a 5-item checklist (see Appendix for form) comprised of *Yes/No* questions about the therapist's or parent's implementation of intervention procedures. To determine agreement for each coded session, trial-by-trial IOA was calculated by dividing the number of checklist items scored as correct by the total number of checklist items and converting the result to a percentage. For example, if observers scored the therapist or parent the same for 4 of the 5 items on the checklist, the trial-by-trial IOA score would be 80% for that session. Table 2 depicts treatment fidelity scores for each participant. Mean treatment fidelity was 88.5% for Luke (range, 75% to 100%), 96.35% for Amy (range, 87.5% to 100%), and 92.31% for Ben (range, 75% to 100%). Across participants, the most common error was failure to provide immediate consequences (i.e., delivered within 3 seconds) following an occurrence of loud speech. Interobserver agreement on treatment fidelity was collected for all treatment fidelity sessions (see Table 3), and agreement was 98.96%.

Chapter 3: Results

Figure 1 depicts participants' frequencies of loud speech per minute as measured by a sound level meter in A-weighted decibels (dBA). Of the four participants who began the study, only three received treatment. The fourth participant (Manuel) was withdrawn from the study by his mother due to changes in the family's schedule. Figure 2 depicts his baseline data across 4 sessions.

During the initial baseline phase for Luke, rates of loud speech varied between 4.6 and 5.0 occurrences per minute ($\mu = 4.84$, $SD = 0.17$) across 5 sessions. With the introduction of the treatment package, rates of loud speech rapidly decreased, varying between 0.2 and 1.2 ($\mu = 0.68$, $SD = 0.37$) before stabilizing after 8 sessions. After reversing to a second baseline phase, rates of loud speech across 5 sessions increased relative to the first treatment phase, but did not return to initial baseline rate levels ($\mu = 2.0$, $SD = 0.24$). When the treatment package was re-introduced, rates of loud speech were similar to those observed in the initial treatment phase ($\mu = 0.37$, $SD = 0.51$), and no occurrences of loud speech were observed in the final three treatment sessions.

During Amy's initial baseline phase, she engaged in loud speech between 5.8 and 7.6 times per minute ($\mu = 6.91$, $SD = 0.56$) across 7 sessions. After implementing the treatment package, rates of loud speech rapidly decreased, varying between 0 and 0.8 ($\mu = 0.28$, $SD = 0.33$) before stabilizing after 5 sessions. When the treatment package was withdrawn, rates of loud speech across 7 sessions ($\mu = 2.49$, $SD = 1.80$) had a steeply increasing trend, with the rate of the final baseline session (i.e., 6 occurrences of loud speech per minute) nearing the mean rate observed during her initial baseline phase.

Upon re-introducing the treatment package, rates of loud speech decreased immediately to near-zero levels ($\mu = 0.08$, $SD = 0.18$), and no occurrences of loud speech were observed in the final three treatment sessions.

During the initial baseline phase for Ben, rates of loud speech varied between 5.2 and 11.8 occurrences per minute ($\mu = 7.31$, $SD = 1.88$) across 9 sessions. With the introduction of the treatment package, rates of loud speech rapidly decreased, varying between 0.2 and 1.5 ($\mu = 0.88$, $SD = 0.58$) before stabilizing after 6 sessions. After withdrawing the treatment package, rates of loud speech were comparable to initial baseline rates ($\mu =6.48$, $SD = 0.69$). When the treatment package was implemented for a second time, rates of loud speech rapidly decreased once again ($\mu = 0.47$, $SD = 0.55$), and no occurrences of loud speech were observed in the final treatment session.

Statistical Analyses

A paired-samples t-test was conducted for each participant to determine whether the difference in mean rates of loud speech per minute in the initial baseline and final treatment phases was statistically significant. The effect size for the treatment, reported as a standard mean difference (SMD) score, was also calculated for each participant. This score was calculated by determining the mean of the initial baseline and final treatment phases and dividing the difference between these scores by standard deviation of the initial baseline phase. The results are interpreted as the number of standard deviations of advantage for a participant receiving treatment, where changes of 0.80 or more standard deviations suggest a large treatment effect.

T-tests

For Luke, there was a statistically significant difference between rates of loud speech per minute during the initial baseline ($\mu = 4.84$, SD = 0.17) and final treatment ($\mu = 0.37$, SD = 0.51) phases; $t(4)=14.37$, $p < 0.0001$.

There was also a statistically significant difference between Amy's rates of loud speech per minute during the initial baseline ($\mu = 6.91$, SD = 0.56) and final treatment ($\mu = 0.08$, SD = 0.18) phases; $t(4)=27.24$, $p < 0.0001$.

Finally, the difference between Ben's rates of loud speech per minute during the initial baseline ($\mu = 7.31$, SD = 1.88) and final treatment ($\mu = 0.47$, SD = 0.55) phases was also statistically significant; $t(5)=12.92$, $p < 0.0001$.

Table 4 depicts t-tests results for each participant. Together, the results of these *t*-tests suggest the decreases in occurrences of loud speech per minute that were observed when the treatment package was implemented are highly unlikely to be the result of random chance.

Effect Size: Standard Mean Difference

The effect size for treatment was greatest for Luke, whose SMD score was 26.29. This score suggests treatment provided more than a 26 standard deviation improvement in Luke's rates of loud speech. For Amy, whose SMD score was 12.20, treatment also has a large effect on rates of loud speech. Finally, although the magnitude of improvement resulting from treatment was lower for Ben (SMD= 3.64), the treatment effect was still considered large. Thus, the treatment package under study yielded a large treatment effect for all participants.

Chapter 4: Discussion

The present study provides preliminary evidence of an effective behavioral treatment for loud speech in children with ASD. All three children who received the treatment, which consisted of an antecedent modification, *in vivo* reminders of behavioral expectations, and a DRO procedure, demonstrated marked decreases in loud speech when the intervention was implemented. Six total demonstrations (2 per participant) of large treatment effects were observed. These results are consistent with the hypothesis that a multi-component behavioral intervention implemented by therapists or caregivers can produce meaningful changes in loud conversational speech for this population.

Although issues with speech loudness have been well documented in individuals with ASD and other developmental disabilities (Sigafoos, Schlosser, O'Reilly, & Lancioni, 2011), descriptions of effective, systematic interventions for this issue have been scarce (Lancioni, Markus, & Behrendt, 1998; Ormand & Mills, in preparation). The paucity of research focused on speech loudness is especially concerning given the importance of prosody to social communication (Green & Tobin, 2009). As such, the current study serves to address the dearth of research in this area by presenting and evaluating a novel behavioral treatment for loud speech with children known to have difficulties regulating their speaking volume during highly preferred activities.

Implications

The outcomes observed in the present study suggest it is possible to decrease rates of loud speech to near-zero levels by consistently implementing a relatively simple combination of behavioral strategies that have been shown to reduce other challenging behaviors with this population (e.g., Brosnan & Healy, 2011; Whitaker, 1996). Importantly, these results also conform to the behavioral principles that underlie the treatment package. Thus, these results extend the application of treatments that are based on sound, behavioral principles to an understudied applied area. Moreover, the present study utilizes a measurement tool (i.e., an application designed for tablets and smartphones) that is inexpensive and readily available to clinicians, teachers, and caregivers, which may increase the feasibility of implementing the intervention in a variety of settings.

In a similar vein, each component of the treatment package under study requires little time and training to implement with high procedural integrity, as evidenced by the high rates of protocol adherence observed for each person (i.e., behavioral therapist or caregiver) conducting treatment. These results suggest that most adults involved in a child's daily care, including parents, teachers, and other clinicians, are likely capable of using the strategies presented herein as part of the child's typical routine to decrease rates of loud speech.

Notably, Luke's loud speech never returned to the rates observed during the initial baseline once the treatment was implemented. This pattern suggests that the failure to independently regulate speech loudness may be a skill deficit rather than a functionally maintained behavior for some individuals. That is, the persistence of loud speech may be attributed to difficulty recognizing the environmental and affective cues that would otherwise serve to punish this behavior, rather than the result of social reinforcement (Sigafoos et al., 2011). Accordingly, it is plausible that some individuals who consistently receive the intervention implemented with participants in the present study can learn to self-regulate their speech loudness over time. Moreover, these individuals may receive more naturalistic reinforcement, such as improved social relationships, as their skills are generalized to less contrived environments. In this case, it might be possible for the intervention to be discontinued without causing a regression to prior rates of loud speech.

Limitations and Future Research

There are several notable limitations to the present study, and each has implications for future investigations of loud speech. First, only three participants ultimately received the treatment package. Although it is not uncommon for single-case studies to include a limited number of participants, it is difficult to discern whether the outcomes observed for these three participants would generalize to other participants, or even other settings, given the small sample of participants receiving treatment. However, the design used to evaluate the treatment package allowed for six total demonstrations of a treatment effect, and large decreases in loud speech were observed during each implementation of the treatment. These outcomes suggest that this treatment package is likely effective when implemented in comparable situations or settings with children whose patterns of loud speech are similar to that of study participants.

One factor that may have influenced the outcomes observed in the present study is the fact that all participants were familiar with DRO procedures due to previous experience with behavioral interventions. Similarly, the therapists and caregiver were also experienced in the implementation of such interventions. Taken together, these history effects may have resulted in more rapid changes in rates of loud speech during treatment phases. Future research should evaluate the present treatment package with children who have little or no experience with behavioral interventions, or with caregivers who have limited experience implementing these interventions, to determine if rapid decreases in loud speech are still observed. With regard to other characteristics that may have made the present sample unique, all participants were highly verbal and motivated to converse with a known adult during social games. As a result, it is unclear whether children who use less language, such as very young children or those who are less socially motivated, would benefit from the treatment presented in this study. It is even possible that this intervention may punish attempts to communicate and engage with others if used with children who are still developing an expressive vocabulary and social approach behaviors. Thus, it may be worthwhile to implement the intervention under study with individuals who differ demographically from the participants who received treatment to determine if similar outcomes are observed with other samples. Additionally, it may be useful to replicate this research in more naturalistic social settings (e.g., including peers) to determine both the social validity and the generalizability of the treatment package under study.

Another important limitation of the present study is the inability to determine which treatment component(s) produced the observed effects as a result of the experimental design. Because all three components of the intervention were implemented simultaneously, it is impossible to know whether a single component in isolation would have produced the same effects. Moreover, if the implementation of only one or two treatment components yielded similar results, it would be more efficient and thus preferable to implement a simpler intervention. In future investigations, it will be important to include component analyses to determine the extent to which each treatment component contributed to the observed decreases in participants' loud speech so that relatively less important components could be omitted in future investigations or applications of the intervention.

For children who exhibit decreases in loud speech as a result of this intervention, future research should also consider conducting a follow-up phase utilizing biofeedback and self-monitoring, which could serve to facilitate generalization and maintenance of appropriate conversational loudness. Finally, as with all research, it is important that the treatment package under study be replicated by independent researchers (i.e., those who were not involved in its development) to reduce the threat of bias.

Table 1.

	# of Intervals of Agreement	Total # of Intervals	% Agreement
Luke	240	240	100.0
Amy	235	240	97.92
Ben	218	228	95.61
Total	693	708	97.88

Interobserver agreement for frequency of participants' loud speech.

Table 2.

	Rater 1 Scoring			Rater 2 Scoring		
	Total Correct	Total Trials	% Integrity	Total Correct	Total Trials	% Integrity
Caregiver 1 (Luke)	177	200	**88.50**	57	64	**89.06**
Therapist 1 (Amy)	185	192	**96.35**	62	64	**96.88**
Therapist 2 (Ben)	192	208	**92.31**	59	64	**92.19**
Total	554	600	**92.33**	178	192	**92.71**

Treatment integrity scores for therapists/caregiver.

Table 3.

	# of Trials of Agreement	Total # of Trials	% Agreement
Caregiver 1 (Luke)	63	64	**98.44**
Therapist 1 (Amy)	64	64	**100.00**
Therapist 2 (Ben)	63	64	**98.44**
Total	190	192	**98.96**

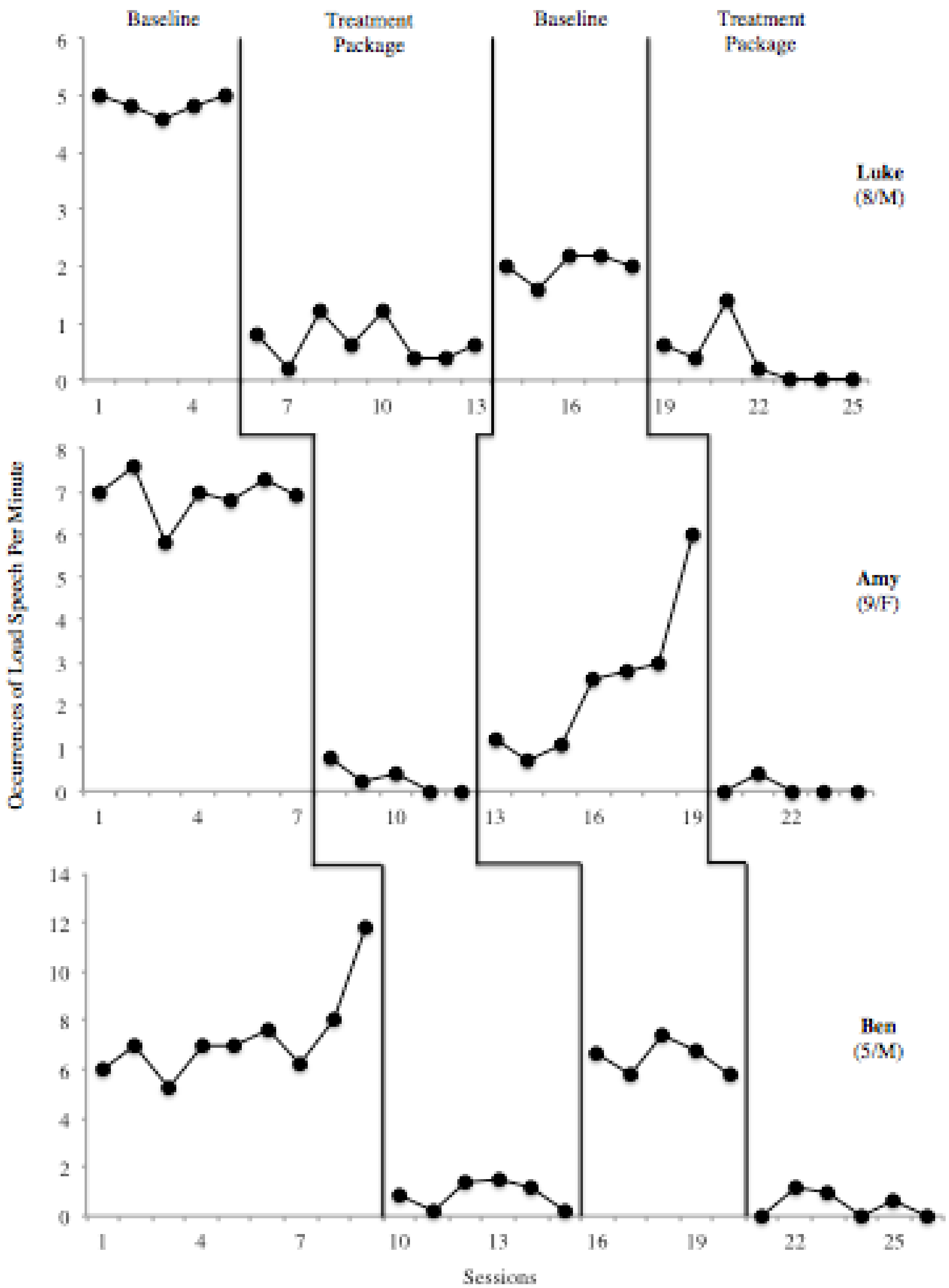
Interobserver agreement for treatment integrity.

Table 4.

Participant	Initial Baseline (*SD*)	Final Treatment (SD)	Degrees of Freedom	*t*-test	*p*-value
Luke	4.84 (*0.17*)	0.37 (*0.51*)	4	14.37	<.0001
Amy	6.91 (*0.56*)	0.08 (*0.18*)	4	27.42	<.0001
Ben	7.31 (*1.88*)	0.47 (*0.55*)	5	12.92	<.0001

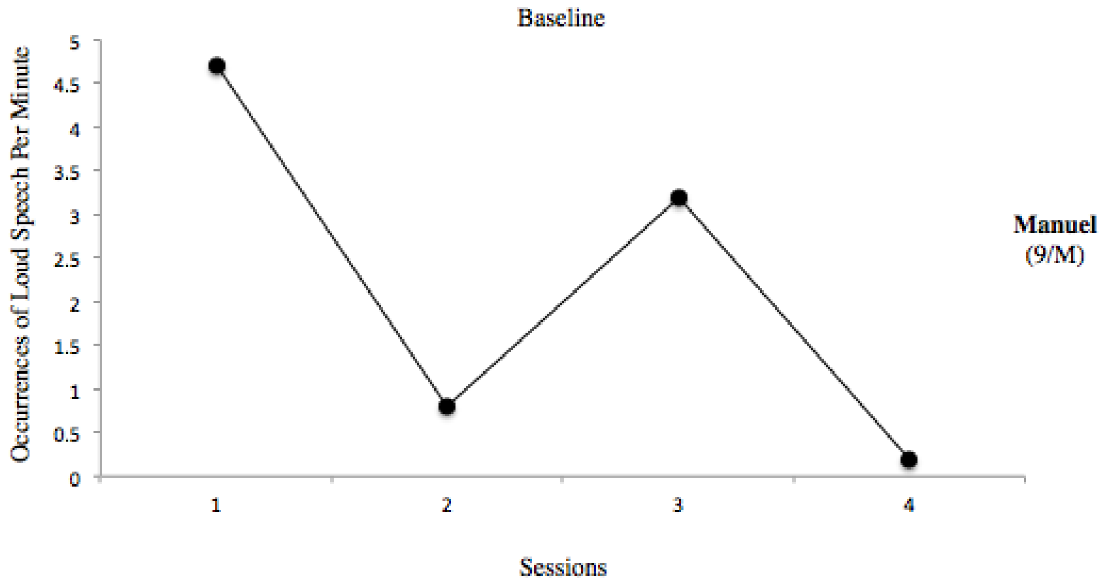
Results of *t*-tests for differences in participants' initial baseline and final treatment rates of loud speech.

Figure 1.



Occurrences of loud speech per minute for participants 1-3.

Figure 2.



Occurrences of loud speech per minute for participant 4.

APPENDIX A

Idiosyncratic Prosody in Autism Spectrum Disorder:

A Review of the Intervention Literature

Over the past 60 years, a large body of scholarly literature has amassed on speech delays, speech impairments, and other idiosyncrasies of speech in individuals with autism spectrum disorders (ASD; for a review, see Matson, Kozlowski, & Matson, 2012). By current estimates, approximately 25-40% of the ASD population is unable to communicate vocally. Further, there is also considerable evidence to suggest that those individuals who do develop speech still tend to have at least some difficulty communicating functionally. Taken together, these patterns of poor social communication contribute largely to the social challenges faces by people with ASD today.

Atypical prosody, such as irregular patterns of stress or monotone speech, appears to be a particularly problematic aspect of communication for this population. Indeed, prosodic speech deficits have been a notable characteristic of ASD since autistic syndrome was first described by Kanner (1943). These deficits can be specific, affecting only certain characteristics of speech or contexts of communication, or relatively global, affecting multiple aspects of speech and occurring during nearly every social exchange (Sigafoos, Schlosser, O'Reilly, & Lancioni, 2011).

Prosody

The term *prosody* is derived from the Greek word *prosōidía*, meaning "song sung to music". In modern linguistics, prosody refers to the patterns of sound used in speech.

The acoustic features of prosody in spoken language include variations in loudness, pitch, rhythm, intonation, and syllable weight (stress). Together, variations in these features convey socially important information and may change or otherwise add to the meaning of speech content (Couper-Kuhlen, 1986).

Disordered expressive prosody appears to be a consistent and chronic speech deficit in individuals with ASD (McCann & Peppé, 2003; Sigafoos et al., 2011). Evidence from multiple studies suggests that individuals with ASD are significantly worse than typically developing controls at perceiving, imitating, and comprehending the meaning of prosodic patterns (Diehl, 2008; Diehl & Paul, 2012). In a similar vein, there is also evidence that individuals with ASD exhibit syllable stresses that are longer in duration than the stresses of their typically developing peers (Diehl & Paul, 2012). Because lower functioning individuals with ASD are less likely to have the verbal abilities necessary for conversational speech, most research examining prosody in ASD has focused on individuals with moderate to high-functioning autism or Asperger's Disorder.

Characteristics. Although the patterns of prosody may vary across languages, the prosodic features of a given language are generally able to convey important information about the speaker or the utterance. This information includes, but is not limited to: the emotional state of the speaker; the presence of irony or sarcasm; the form of utterance (e.g., statement, command, question); and emphasis, contrast, and focus (Fernández & Cairns, 2011). Clearly, the ability to perceive and correctly interpret prosodic characteristics (e.g., lexical stress) is a crucial aspect of language development (Ballard, Djaja, Arciuli, James, & van Doorn, 2011). In fact, evidence suggests that infant-directed prosody may actually be requisite for infants to pair sounds with meanings (Estes & Hurley, 2013).

Rhythm and rate. In terms of prosody, rhythm refers to the meter, pace, or tempo of speech. In spoken language, grammatical units are typically ordered and spoken in patterns that are synchronous and predictable. Because the rhythmic coordination of interactions between humans can be considered essential to communication and important to social and emotional connection, atypical rhythm of speech can lead to disrupted engagement and decreased feelings of connection between speaker and listener (Borrie, 2014).

Patterns of idiosyncratic rhythm can have widely varying topographies in individuals with ASD. For example, some individuals may speak at a much slower rate than their typically developing peers (Beltaxe & Simmons, 1977), which may make it more difficult for them to establish a comfortable rhythm of speech and effectively engage the listener. On the other hand, there is also evidence that individuals with Asperger's Syndrome (AS) may speak much quicker than others, including both their neurotypical counterparts and individuals diagnosed with high-functioning autism (HFA; Shriberg, Paul, McSweeny, Klin, Cohen, & Volkmar, 2001). In still other cases of idiosyncratic rhythm, some individuals with ASD have been described as speaking in an overly animated or "sing-song" rhythm (Charlop, Dennis, Carpenter, & Greenberg, 2010).

Stress. As a prosodic characteristic of speech, stress refers to the emphasis placed on a grammatical unit, such as a syllable in a word, or a word in a sentence or phrase. Stress is often used to reflect the relative importance of each word in a sentence, and can be conveyed by increased loudness or vowel length. Stressed syllables are defined by their higher pitch, longer duration, and increased loudness relative to other syllables (Bellon-Harn, Harn, & Watson, 2007). When only one word is stressed, the speaker conveys that the stressed word is the focus of their utterance. As a result, the use of stress can provide clarity in the case of misunderstanding or ambiguity (Price, Ostendorf, Shattuck-Hufnagel, & Fong, 1991).

In an experimental investigation of the relation between lexical stress and prosody in adolescents with high-functioning autism (HFA), Grossman, Bemis, Skwerer, and Tager-Flusberg (2010) found that participants with HFA were as proficient as typically developing controls at perceiving lexical stress. Despite being capable of differentiating lexical stress patterns, however, the authors found that the HFA group had atypical lexical prosody production. More specifically, the lexical stress of children with HFA tended to be longer in duration than the stress of controls.

Pitch and intonation. Acoustically, pitch can be conceptualized as the degree or highness or lowness of a tone. Because pitch does not convey much semantic information in Indo-European languages (e.g., English), changes in pitch usually do not change the meaning of spoken language. In tonal languages (e.g., Mandarin Chinese), however, changes in pitch can change the meaning of a word. Intonation, or tone of voice, refers to pattern or melody of pitch changes in speech. Variations in intonation can be used to convey sentence type (e.g., question, statement, command) as well as the emotional state of the speaker.

Israeli Hebrew (IH) can be considered a tonal language, as tone can be used in IH to provide semantic distinctions in communication. After conducting a thorough analysis of prosody in 20 Hebrew-speaking children (10 with ASD, 10 typically developing), Green and Tobin (2009) found that children with ASD used high pitch accents more frequently than their typically developing peers. Additionally, the authors also found that children in the ASD group exhibited a relatively limited repertoire of edge tone patterns, and were overly repetitive in their use of these patterns. Taken together, the monotonous accent and the repetitiveness of edge tones resulted in what the authors describe as "a stiff sounding prosody" in the ASD group. In contrast, the prosodic patterns of typically developing participants were described as more diverse and flexible (Green & Tobin, 2009).

In another examination of pitch and intonation, Nakai, Takashima, Takiguchi, and Takada (2014) conducted a quantitative acoustic analysis of speech in children with ASD. Results from this study indicated that monotonous speech was more common for children with ASD than for their typically developing peers. Moreover, the authors discovered that children who exhibited more variable pitch in their speech also exhibited more reciprocal social interactions than children who were monotonous, even after controlling for cognitive ability (Nakai et al., 2014).

In contrast to evidence suggesting that children with ASD are more likely than their typical peers to have monotonous speech (Green & Tobin, 2009; Nakai et al., 2014), other evidence suggests that tone and pitch variability may actually be greater than average in this population. Studies by Bonneh, Levanon, Dean-Pardo, and Adini (2011) as well as Nadig and Shaw (2012) found that children with ASD demonstrated a significantly larger pitch range and variability than typically developing controls. According to study authors, this evidence could reflect impaired processing of auditory stimuli, or disorder in the mechanisms that control pitch. If the mechanisms controlling pitch do not function properly in ASD, this factor could help to explain why study results from Bonneh and colleagues (2011) were inconsistent with previous findings of monotonous speech. In this case, it is possible that some individuals with ASD would exhibit monotonous speech, whereas others, especially those with high pitch frequency, might exhibit more variable pitch and tone (Diehl & Paul, 2009).

Loudness. In terms of speech, loudness refers to the intensity or volume of the sound produced. For individuals with ASD and other developmental disabilities, issues with speech loudness can manifest in several different ways (e.g., Pronovost, Wakstein, & Wakstein, 1966), with each having unique effects on social communication. There is evidence to suggest that some individuals with ASD speak with insufficient intensity (i.e., too quietly); in contrast, others speak with excessive intensity (i.e., too loudly). Inconsistent vocal intensity, which is characterized by variable loudness, is also common among this population and can have unintended effects on perceptions of intonation, such that a listener may perceived a word or phrase as stressed even if the speaker had not intended to convey this emphasis (Bone et al., 2014).

One potential explanation for the overly quiet speech sometimes observed in ASD is that hyperacusis, or abnormal acuteness of hearing, is more common among individuals with ASD than among the neurotypical population (Khalfa, Bruneau, Roge, Georgieff, Veuillet, & Adrien et al., 2004). For those who speak atypically loudly, their own speech volume may seem comparable to the loudness of others. On the other hand, speakers with atypically quiet speech may perceive the speech of others as too loud. Another possible explanation for the inappropriate patterns of speech loudness common among individuals with ASD is that the well-documented deficits in social perspective taking and theory of mind observed in this population may resultantly cause these individuals to be unaware of their vocal loudness relative to that of others.

Growing evidence suggests inappropriate or atypical speech volume can be an urgently problematic area of social communication for both children and adults with ASD, as this issue may make it very difficult for someone to work or be educated with the mainstream population. Thus, atypical volume appears to be an important area for speech and language intervention (Koegel & Frea, 1993; Lancioni, Markus, & Berhendt, 1998; Ozdemir, 2008).

Impact of Prosody on Social Communication

Just as patterns of phonology vary across languages, so, too, do patterns of prosody. As mentioned previously, changes in intonation may influence the meaning of a word in tonal languages. As a result, certain characteristics of prosody may be more important in some languages than in others. Atypical or incorrect use of prosody is a common cause of misunderstandings between native speakers and second-language learners.

Because prosody conveys socially meaningful information, it follows logically that difficulty using or understanding certain prosodic elements would almost certainly create challenges during social interactions. Prosody is typically absent in written language, which can sometimes lead to misunderstandings of the language content. Those individuals who are unable to distinguish the prosodic subtleties of speech may misinterpret the meaning of the spoken content, as is often the case with dry, complex, non-literal humor (e.g., sarcasm, irony). Indeed, a comedian's "delivery" essentially amounts to his or her use of prosody.

Problems with loudness of speech can have a profound effect on social interactions. Some people may find it difficult or frustrating to converse with someone who speaks in a whisper, or may misunderstand the speaker's intended message. Similarly, others may find it embarrassing to converse with someone whose voice is inappropriately loud for a given setting. Individuals with hyperacusis may even find it painful to listen to an overly loud speaker. Because children with ASD may be more likely to socialize and receive specialized instruction together than with their neurotypical peers, it seems concerning that children with hyperacusis might find it punishing or otherwise unpleasant to converse with children whose conversational volume is louder than average (Khalfa et al., 2004; Lucker, 2013). Thus, inappropriate conversational volume may be detrimental to children's socialization even in contrived intervention settings.

Emotional prosody refers specifically to the expression of emotions using prosodic components of speech (Pittham & Scherer, 1993). In the late nineteenth century, Charles Darwin theorized that emotional prosody likely predated the evolution of human language, as he had observed a variety of other animal species using prosody in communication: "Even monkeys express strong feelings in different tones— anger and impatience by low, fear and pain by high notes." (Darwin, 1871). Thus, from an evolutionary perspective, it appears that understanding and using prosody may be essential to effective social communication.

For individuals with ASD, it is apparent that recognition of emotional-prosodic meanings may be an especially challenging aspect of language development and social communication. In a study by Van Lancker, Cornelius, and Kreiman (1989), children with ASD and schizophrenia were asked to label four emotional intonations used in speech, and their performances were compared to those of age-matched controls. Unlike their typically developing peers in the control group, older children with ASD were unable to reliably identify the emotional intonations presented to them. However, it is important to note that younger children with ASD performed no worse at this task than their age-matched peers in the control group, which suggests that the ability to recognize and label emotional states based on prosodic features of speech may not emerge or become well-developed until middle childhood or later (Van Lancker, Cornelius, & Kreiman, 1989).

In a similar study of emotional prosody, Lindner and Rosén (2006) compared the abilities of individuals with Asperger's Syndrome (AS) to decode emotion through facial expression, prosody, and language content to the abilities of their typically developing peers. Consistent with other research in the area, study authors found evidence that individuals in the AS group had more difficulty identifying emotions through prosody than those in the control group. Based on this evidence, the authors suggest that individuals with AS may become over-reliant on verbal content over time as a compensatory strategy for their deficits interpreting prosodic content (Lindner & Rosén, 2006).

Not surprisingly, research suggests that idiosyncratic prosody may negatively affect social perceptions of the speaker (Mesibov, 1992; Paul et al., 2005; Shriberg & Widder, 1990). In one examination, Page and Balloun (1978) conducted an experiment to determine how a speaker's volume affected listener's ratings of the speaker across various domains. As hypothesized, evidence from this study indicated that speakers who were louder than average were perceived as most aggressive, but also lacking in self-assurance. Similarly, Paul and colleagues (2005) found an association between sentential stress, nasality, and ratings of participants' sociability and communication skills, such that participants with atypical setential stress and hypernasality were rated as having poorer sociability and communication skills than participants with typical speech prosody.

Development of Prosody

Speech development is an inherently social process. There is considerable evidence that children who are spoken to more frequently, and thus experience more interactions with language, tend to develop better language abilities than similar children who are spoken to less. In turn, children who have greater language abilities tend to evoke more interactional language from their environment (e.g., by making requests, commenting, and asking questions) than children with less developed language abilities. Over time, this cycle of language development can create an ever-widening gap in children's communicative abilities, in which children with the best language skills receive more exposure to language than do children with poorer language skills. Unfortunately, children with ASD may be some of the most likely to fall victim to this effect.

To better understand the social communication deficits that characterize ASD, Warlaumont, Richards, Gilkerson, and Oller (2014) examined the transactional patterns of communication between adults and children with or without ASD. Study investigators hypothesized that socially communicative vocalizations (i.e., "speech-related vocalizations"), in contrast to non speech-related vocalizations such as vocal stereotypy, were more evocative of a corresponding speech-related vocalization from an adult. Not surprisingly, authors indeed found evidence that speech-related vocalizations were more likely than non-communicative vocalizations to be reinforced by a social response. In turn, they also discovered that a child's vocalization is more likely to be speech-related if his or her previous speech related vocalization received a social response, thus creating a social feedback loop (Warlaumont et al., 2014). According to the authors, this feedback loop has far-reaching effects on speech development, such that the speech of typically developing children will continuously be reinforced, shaped, and expanded over time, whereas the speech of children with ASD will become increasingly limited in comparison to their peers.

Because prosody is a linguistic device that indicates the relationship between grammatical units of a sentence, a young child's understanding of prosodic characteristics of speech is an important aspect of learning of his or her native language (Lahey, 1974). As such, children who have difficulty understanding prosody may experience some degree of speech delays, including deficits in their own use of prosody. If children with ASD are exposed to fewer communicative exchanges early in life than their typically developing peers, it is apparent that these children will have greater difficulty learning and understanding some of the more nuanced aspects of language and speech, including prosody.

What causes prosody deficits in ASD?

There is much disagreement among researchers about the root cause of idiosyncratic prosody in ASD. Studies of emotional speech awareness in children with ASD suggest that these individuals may not be "tuned in" to the affective states of others, as evidenced by their failure to respond appropriately to prosodic speech characteristics reflecting high-alert states (e.g., increased loudness, excited tone; Yanushevskaya, Gobl, & Chasaide, 2013). When added to the other literature on prosody in autism, these results suggest that difficulties using prosodic speech in this population may actually be the result of more a primary difficulty in processing the prosodic information they receive from their environments. In turn, deficits in receptive processing of prosodic speech may be related to an even broader, over-arching deficit in auditory processing (O'Connor, 2012). Logically, it seems that children who have difficulty accurately processing and interpreting auditory information would also likely have difficulty distinguishing prosodic features of speech, and that children who are unable to accurately distinguish prosodic features would likely be less proficient in their own use of prosody.

There are several potential explanations for the prosodic deficits seen in ASD. From a synthesis of the evidence, it is appears that children with ASD may have difficulty: (1) attending to and distinguishing prosodic features of speech in others, (2) receiving the social and emotional information communicated via prosody, (3) understanding the social importance of prosodic speech, and (4) using prosody to enhance or add meaning to social communication.

Interventions for Atypical Prosody in ASD

Similar to the treatment of other behavioral excesses and deficits seen in ASD, research suggests that behavioral interventions (e.g., discrete trial training, modeling, shaping) may be the best approach to improving prosodic speech deficits in this population (Sigafoos et al., 2011). Despite the wealth of evidence that deficits in prosodic speech are common for individuals with ASD, however, there is a relative dearth of literature detailing evidence-based interventions for these chronic and pervasive language deficits. Several descriptive studies on prosodic speech in ASD have suggested there is an urgent need for more intervention research in this area (e.g., Peppé, McCann, Gibbon, O'Hare, & Rutherford, 2007). The paucity of research on interventions for atypical prosody is especially concerning given the importance of prosody to effective social communication, as well as the influence it has on social perceptions of the speaker. Thus, the purpose of the present review is to identify and summarize the extant research in this area, and to evaluate the design, methods, and conclusions presented in the identified articles based on current methodological standards for single-case research.

Method

Studies were included in the present review based on the following criteria: the study (a) included participants with autism/ASD, developmental disabilities, or described as having autistic features, (b) described and/or evaluated an intervention for atypical prosodic speech in this population, and (c) was published in English between 1970 and 2014 in a peer-reviewed journal. Although earlier studies (e.g., published before 1990) may have employed methods considered aversive by current treatment standards, these articles were included in this review to illustrate the true dearth of intervention research in this area. Electronic searches were conducted using ERIC, PsycINFO, and MEDLINE databases using the search terms “autism”, “prosody”, and “treatment” or “intervention”. Each article that was identified through these searches was read in its entirety by the primary investigator of the present review. To identify a more complete body of literature, follow up searches were conducted by hand using the reference sections of the articles identified through electronic searches.

Results

Six studies were identified for inclusion in this review; these studies are summarized below in Table 1. After compiling these studies, each was coded for research design, treatment approach, intervention type, behavioral topography, and the prosodic speech component(s) targeted for intervention. Five of the six studies in the present review used single-case research designs to assess treatment effects; the remaining study (Lim, 2010) had a much larger sample size (n = 50) and used an independent samples t-test to assess treatment effects. Notably, Lin's (2010) study is also the only in this review to find limited treatment effects on prosody.

Table 5. Summary of interventions for atypical prosody in ASD.

Study Author(s)	*Prosodic Characteristic Targeted*	*Participant Age/Gender*	*Design*	*Theoretical Orientation of Intervention*	*Category*	*Intervention*	*Behavioral Topography*
Bellon-Harn, Harn, & Watson (2007)	Rhythm, stress	8/M	AB	Psychosocial, behavioral	Natural environment training (NET)	"Interactive approach to language"; (Modeling, shaping)	Lengthened stress duration resulting in idiosyncratic rhythm
Charlop, Dennis, Carpenter, & Greenberg (2010)	Intonation, stress	11/M 8/M 7/M	Multiple baseline across participants	Behavioral	Modeling	Video modeling	Poor intonation, monotone speech
Koegel & Frea (1993)	Loudness	13/M	Multiple baseline across participants and behaviors	Behavioral	Differential reinforcement of other behavior (DRO)	Self-monitoring	Loud conversational voice when excited
Lancioni, Markus, & Behrendt (1998)	Loudness	20/M	ABAB	Behavioral	Differential reinforcement of other behavior (DRO)	Biofeedback (vibrating watch)	Loud voice at work
Lim (2010)	Pitch, loudness	50 participants ages 3-5 y.o.	AB	Developmental, behavioral	Music Therapy	"Developmental Speech and Language Training Through Music" (Video modeling)	Poor general prosody
Ozdemir (2008)	Loudness	9/M	Multiple baseline across participants	Behavioral	Antecedent modification	Social Story	Loud voice in classroom

Bellon-Harn, Harn, and Watson (2007)

In this case study, the authors describe an interactive, naturalistic intervention for prosody in an 8-year-old boy with high-functioning autism (HFA). Despite receiving a language intervention for three years and making great gains in language, the participant's prosodic speech remained "markedly impaired". The treatment for this child included theme-based (i.e., science), child-led therapy sessions that created natural opportunities for the child to engage in turn-taking conversation with an adult. Using an AB design, the authors assessed treatment effects on the target behavior of lengthened syllable duration, which appeared to create the idiosyncratic patterns of stress and rhythm exhibited by the child. Results suggest a significant decrease in syllable duration following the implementation of the intervention, and in turn, improvements in the stress and rhythm of the child's speech.

Charlop, Dennis, Carpenter, and Greenberg (2010)

In a study by Charlop and colleagues (2010), a multiple baseline across-participants design was used to assess test the effects of a video modeling intervention on the intonation of three boys (ages 7-11) with autism. Concerning behavioral topography, two of the children were described as having monotone speech, whereas the third child was described as soft-spoken with a "sing-song" tone voice. Treatment for each child consisted of an individualized video depicting a brief interactional scenario (e.g., a child seeing a preferred toy, a child making a basket) in which a verbal comment might be appropriate. These scenarios were acted out between two familiar adults, including one adult acting as a child model, with focus placed on the intonation, gesture, and facial expression appropriate for the given scenario. Results indicate that intonation was improved for all three participants, and these outcomes were maintained to a moderate degree over time.

Koegel and Frea (1993)

In a study of self-management, Koegel and Frea (1993) examined the acquisition of social communicative behaviors in two boys with autism, including one child who spoke too loudly when he became excited about a conversational topic. More specifically, a multiple baseline across participants design was used to assess the effects of a self-monitoring procedure, which included a differential reinforcement of alternative behavior (DRA) component, on this participants' conversational volume. Inter-observer agreement (IOA) data from blind observers indicated that the child was able to reliably monitor his own conversational volume, and that the self-monitoring intervention resulted in significant increases in his use of appropriate loudness. Follow-up probes conducted after the intervention was faded suggest that these positive results were maintained over time, and even generalized to untreated behaviors.

Lancioni, Markus, and Behrendt (1998)

In a study examining the treatment utility of biofeedback in individuals with ASD, Lancioni, Markus, and Behrendt (1998) investigated the effect of a portable vibratory-feedback device on the vocal loudness of a man with intellectual disability and autistic-like features. Unlike the previously described studies, this study used an ABAB design to compare treatment effects to baseline data of the participant's behavior. Consistent with predictions, study authors found that the vibratory device was rapidly effective at reducing excessive vocal loudness when paired with contingent reinforcement. The results of this study provide important evidence that individuals with low-functioning ASD can respond to biofeedback and learn to regulate their own social behavior.

Lim (2010)

Unique to the studies included in the present review, Lim's (2010) study compared the effects of two different video modeling interventions for prosody in children with ASD. To test the impact of music training on atypical prosodic speech and other deficits of speech production, fifty children (ages 3 – 5) with ASD were randomly assigned to one of three conditions: speech therapy (n = 18), music therapy (n = 18), or control (n = 14). Results indicate that children in either treatment group tended to show greater improvements in prosodic speech, including pitch accent, length of vowel sounds (duration), and intensity (loudness of speech), than children in the control group. However, the data also suggest that music training is no better than speech training at improving prosodic speech, which is inconsistent with predicted results. Therefore, it is unclear whether music therapy is a desirable choice for practitioners designing interventions for prosodic speech in children with ASD.

Ozdemir (2008)

The final study that met criteria for inclusion in the present review examined the effectiveness of Social Stories on decreasing disruptive behaviors of children with autism, including a 9-year-old boy whose presenting problem was an inappropriately loud voice in the classroom. In this case study, a single-subject, multiple baseline design across participants was used to assess the effects of the intervention. Consistent with study hypotheses, study investigators found evidence that Social Stories may be an effective treatment for the disruptive classroom behavior, including vocal loudness. The results of this study are consistent with other examinations of behavioral treatments for atypical loudness in ASD, and suggest that behavioral interventions may be the most appropriate and effective for this population.

Discussion

The present review aimed to identify and categorize the extant empirical research on interventions for idiosyncratic prosody in ASD. Based on the limited number of scholarly articles that have been published in this area since 1970, it appears that the well-documented prosodic deficits experienced by individuals with ASD are not being adequately addressed and treated with evidence-based interventions. This absence of intervention research is especially concerning given the decades of descriptive evidence on prosodic deficits in ASD, and the critical importance of prosodic speech to natural and effective social communication.

Of the six studies included in this review, four described interventions that were rooted in behavioral theory (Charlop, Dennis, Carpenter, and Greenberg, 2010; Koegel & Frea, 1993; Lancioni, Markus, & Behrendt, 1998; Ozdemir, 2008). The remaining two studies appeared to be rooted in developmental (Lim, 2010) and psychosocial theories (Bellon-Harn, Harn, & Watson, 2007), but also utilized behavioral strategies (i.e., modeling, prompting, video modeling) as major components of their interventions. Taken together, this breakdown suggests that behavioral interventions are the preferred approached to the treatment of atypical prosody in ASD, which is consistent with myriad evidence supporting the use of behavioral interventions with this population.

Of the six interventions included in this review, loudness was the most of targeted prosodic speech characteristics. Moreover, pitch, stress, and rhythm were each only represented as the target behavior in a single article. Based on these observations, it appears that atypical loudness may be the most commonly treated prosodic deficit among individuals with ASD. It is possible that speech characteristics such as pitch and rhythm, although important to conversational speech, are simply less urgent areas for intervention. Because English is not a tonal language, idiosyncrasies in pitch do not change meaning; similarly, idiosyncrasies in stress and rhythm may make a speaker sound less natural, but appear less important to functional communication than to social perceptions. Thus, underrepresentation of some prosodic features within the intervention literature may be related to the limited *necessity* of those particular skills in every day life.

Limitations

The present study has several limitations that are worth consideration. To begin with, one published article describing an intervention for atypical loudness was not included in the review because it was unavailable in English. It is possible that this article contributes greatly to the treatment literature on prosodic speech in this population, and that its omission leaves an important void in the understanding facilitated by the present review. Similarly, it is possible that other developmental and behavioral speech interventions may be effective in the treatment of idiosyncratic prosody, but the appropriateness of their use for treating prosodic deficits has not been confirmed in the scientific literature. Clearly, more research is warranted in this area. In the future, researchers should aim to address this gap in the literature by describing and evaluating new and existing approaches to the treatment of prosodic deficits, especially for the prosodic skills that are most important to functional communication.

Implications

The present review of the literature contributes to the understanding and treatment of idiosyncratic prosody in ASD, and has a number of important implications for research and clinical practice. Although difficulties with prosodic speech are a common and well-documented problem for this population, there is very limited empirical research describing effective interventions for the prosodic deficits seen in ASD. For practitioners who treat ASD, the present review may provide a summative glimpse at the interventions that have already been tried with some success. It is likely that strong interventions for these speech issues could greatly improve social communication for individuals with ASD, which would in turn reduce their degree of functional impairment and improve their social relationships.

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APPENDIX B

Treatment Integrity Rating Form

Observer:

Date:

Study phase/session #:

☑ = behavior was observed
☒ = behavior was not observed

- ☐ 1. Therapist provided participant with verbal reminder of rules about voice loudness prior to entering the target setting.

- ☐ 2. Therapist provided participant with verbal reminder of reinforcement he/she would receive for complying with rules about voice loudness in the target setting.

- ☐ 3. Therapist modeled appropriate voice loudness throughout the entire study session.

- ☐ 4. Therapist remained engaged with and responsive to the participant throughout the entire study session.

- ☐ 5. Therapist provided verbal prompts when necessary to facilitate conversation with the participant.

- ☐ 6. Therapist allowed to child to choose and lead the play activity during the study session.

- ☐ 7. Therapist provided participant with verbal reminder of rules about voice loudness upon each occurrence of the target behavior.

- ☐ 8. Therapist delivered consequences (verbal and visual reminders) within 3 seconds of being instructed to do so by the study investigator.

Treatment Integrity Score = ___/8

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